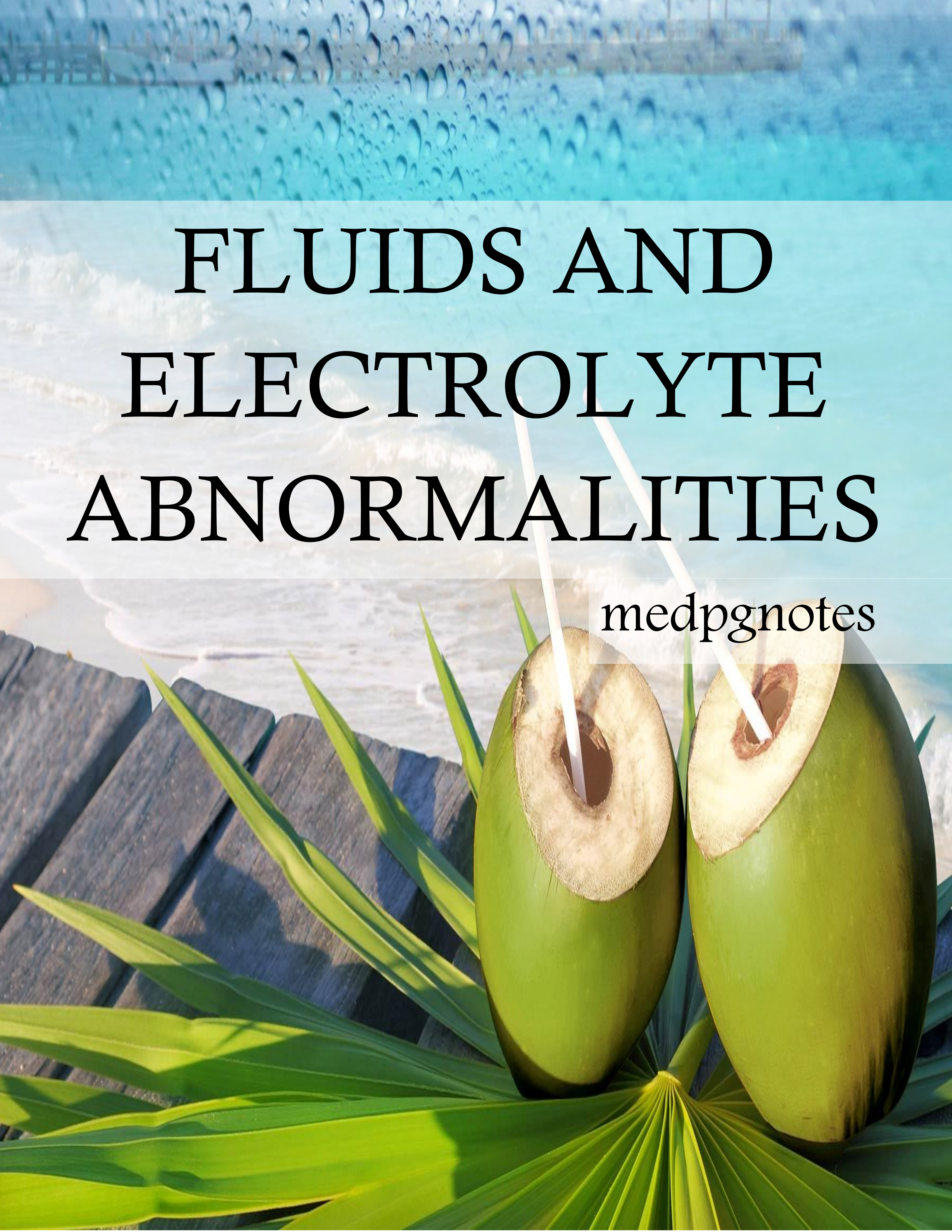
FLUIDS AND ELECTROLYTE ABNORMALITIES

medpgnotes



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FLUIDS AND ELECTROLYTE ABNORMALITIES

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KEY TO THIS DOCUMENT

Text in normal font – Must read point. Asked in any previous medical entrance examinations

Text in bold font – Point from Harrison's text book of internal medicine 18th edition

Text in italic font – Can be read if you are thorough with above two.

BLOOD TRANSFUSION

BLOOD GROUPING

Gene determining ABO blood group	*Chromosome 9p*
Universal blood group donor	O negative
ABO blood group antigen	Found of RBC membrane, Glycoprotein in nature, Highly immunogenic
ABO blood group system remains most important in clinical medicine because	ABO antibodies are invariably present in plasma when person's RBC lack the corresponding antigen
Genotype of a person with blood group A should be	AO
Deoxysugar characteristic of blood group polysaccharide	Fucose
ABO antigen NOT seen in	CSF
Both parents A group	Children A or O
ABO incompatibility occurs when mothers blood group is	O
Bombay Blood Group	Lack of other Blood Group Antigens (only H)
Kell antigen on	**Chromosome X**
MC blood group in Indian people	O
Diagnosis of ABO incompatibility can NOT be made from	CSF
ABO antigen NOT found in	CSF
NOT true about blood grouping	It is the method to conclusively fix paternity
ABO incompatibility is NOT seen with	Cryoprecipitate
MC cause of incompatibility in pretransfusion screening	*Lewis system*

BLOOD COMPONENTS

Blood product least likely to carry infectious disease	*Red blood cells*
Cryoprecipitate is stored at	Minus 40 degrees
Cryoprecipitate is rich source of	VIII
Cryoprecipitate is rich in	Fibrinogen I, Factor VIII, Von willebrand factor
Cryoprecipitate does NOT contain	Antithrombin
Cryoprecipitate does NOT contain	Factor IX
Cryoprecipitate does NOT contain	Factor IX
Cross matching NOT required for transfusion of	Cryoprecipitate
Which blood fraction is stored at -40*C	Cryoprecipitate
Shelf Life of CPD	28 days
Blood stored in CPDA for	35 days
Stored plasma is deficient in	Factor 5 and 8
NOT true about FFP	Used as volume expander
In cholecystectomy, FFP is given	Just before operation
Uses of fresh frozen plasma	*Antithrombin III deficiency, massive transfusion bleeding diathesis, reversal of*

	warfarin effect
Volume of one unit of cryoprecipitate	*10-15 ml*
Volume of one unit of packed RBC	*300 ml*
Volume of one unit of platelet concentrate	*50 ml*
One unit of platelet concentrate increases platelet count by	*10,000*
Maximum lifespan of transfused RBC	60 days
RBC stored in blood bank	Decreased 2,3 DPG, Decreased Na+, Decreased pH, Increased K+
Blood platelets in stored blood do not remain functional after	24 hours
Stored blood	Decrease in ATP, INCREASE in K+, decrease in Na+
IMP is added to stored blood to act as source of	*2,3 DPG*
Platelets can be stored at	20-24*C for 5 days
Refrigerated blood stored up to 48 hours before transfusion can destroy	Treponema pallidum
Irradiation of blood products	*Prevent donor white cells from proliferating in recipient's body*
Cryoprotectant	*Dimethylsulfoxide*

BLOOD TRANSFUSION

Reverse type of pre blood transfusion testing	*To detect isoagglutinin in recipient*
Best investigation done immediately to confirm non matched blood transfusion	Direct coomb's test
Better indicator for need of blood transfusion	Hematocrit
Blood transfusion	Antigen D determines Rh positivity, febrile reaction is due to HLA antigen
Massive blood transfusion defines	Whole blood volume
One unit of fresh blood rises Hb% concentration by	1 gm%
Rh negative individual who is transfused for first time with Rh positive blood is likely to developed	*Delayed transfusion reaction*
Test for non mismatched blood transfusion	*Antibodies in recipient serum*
Indication for calcium replacement after transfusion	*Blood given at faster rate, blood transfusion given to a patient suffering from liver disease, neonates requiring transfusion*

COMPLICATIONS OF BLOOD TRANSFUSION

MC transfusion reaction	Febrile Non hemolytic reaction

Acute hemolytic blood transfusion	Complement mediated hemolysis
Transfusion associated lung injury	*Within 6 hours of transfusion*
Most probable reason for transfusion associated hepatitis	Chronic carriers negative for HbsAg
Transfusion related acute lung injury is triggered by	Anti HLA antibodies
ABO transfusion reaction	Type II reaction
Mismatched blood transfusion in an anesthetic patient present as	Hypotension and bleeding from wound site
Intra operative mismatch blood transfusion	Excessive bleeding, hypotension, bronchospasm, rash
Massive blood transfusion results in	Hyperkalemia, DIC, Hypothermia, Thrombocytopenia
Complication likely to result after several units of blood transfusion	Metabolic alkalosis
Massive transfusion in previous healthy adult male can cause hemorrhage due to	Dilutional thrombocytopenia
NOT a complication of massive blood transfusion	Hypokalemia
NOT a complication of Massive blood transfusion	Metabolic acidosis
NOT a complication of Massive blood transfusion	Hypokalemia, Hypercalcemia
NOT a complication of blood transfusion	Hypokalemia
FALSE about anaphylactic blood transfusion	Occurs in IgG deficient
Treatment of transfusion siderosis	Desferiprone
Drug for Hemolytic transfusion reaction	Steroids
Transfusion transmitted virus	HBV,HTLV I, CMV, Toxoplasma, Syphilis
MC virus associated with Transfusion Hepatitis	HCV
Blood transfusion does NOT transmit	Leukemia
NOT transmitted via blood transfusion	Dengue
NOT transmitted by blood transfusion	EBV
Post transfusion infection is commonly due to	HCV

ELECTROLYTE ABNORMALITIES

ANION GAP

Anion gap	{Na+ + K+} - {HCO3- + Cl-}
Normal anion gap	12 - 16 mEq/L
Anion gap is mostly due to	Protein
Urinary anion gap is an indication for excretion of	NH4+ ion
Increased anion gap	Lactic acidosis, DKA, Cardiac failure
Increased anion gap is NOT seen in	Diarrhea
NOT associated with increased anion gap	Glue sniffing
NOT a cause of Normal anion gap	Aspirin overdose

GENERAL FEATURES OF ACID BASE DISORDERS

Acid base disorder	pH is determined by PCO2 and HCO3-, metabolic acidosis is compensated by decreased PCO2, buffering may be intra and extracellular
Important role in regulation of acid base balance in neonates	*Kidney*

ABG pH 7.2 raised pCO2,decreased HCO3	Respiratory and metabolic acidosis
pH 7.58 pCO2 23 mm Hg,pO2 300mm hg oxygen saturation 60%	Ventilator malfunction
Persistent vomiting most likely cause	Acidic urine, hypochloremia
NOT true about acid base disorder	Respiratory acidosis is compensated by decreased HCO3-

INTERPRETATION OF ABG VALUES

pH < 3.5	Acidosis
pH > 3.5	Alkalosis
Normal bicarbonate	22 - 30 mEq/L
Primary change in bicarbonate	Metabolic
If in acidosis, HCO3 decreased, problem is	Metabolic
Primary change in CO2	Respiratory
Normal PCO2	35- 45 mm Hg
In acidosis, CO2 increased, problem	Respiratory
Plots acid base characteristics of arterial blood and helpful in clinical situations	Siggard Anderson normogram
Plasma Osmolality	$2 * [\mathrm{Na}] + \frac{\mathrm{Glucose}}{18} + \frac{\mathrm{BUN}}{2.8}$
Plasma osmolality of a child with Na+ 125 mEq/L, glucose of 108 mg/dl, BUN of 140 mg/dl	306 mOsm/kg

METABOLIC ACIDOSIS

Normal anion gap metabolic acidosis	Cholera
Normal anion gap metabolic acidosis	Diarrhea, Cholera, Ureterosigmoidostomy, Renal tubular acidosis, Mineralocorticoid deficiency
High anion gap metabolic acidosis	Diabetic ketoacidosis, Lactic acidosis, Methyl alcohol poisoning, Salicylate poisoning, Ethylene glycol poisoning, Toxin ingestion, Acute and chronic renal failure
Lactic acidosis is seen in	Methanol poisoning, Circulatory failure, Respiratory failure
NOT associated with increased anion gap metabolic acidosis	COPD
NOT a cause of increased anion gap acidosis	Glue sniffing
Pathology in metabolic acidosis	*NH3 is actively secreted in collecting duct*
Acute metabolic acidosis	Decreased urinary K+ excretion
Amount of bicarbonate given in metabolic acidosis	1/2 * body weight * (desired HCO3 (24) - measured HCO3)
A child with diarrhea, deep rapid inspiration	Metabolic acidosis
Blood pH is decreased in	Uremia
A child presents with diarrhea and peripheral circulatory failure and pH is 7.PCO2 is 15 mm Hg. Most appropriate therapy	Bolus of ringer lactate
Electrolyte abnormality after ureterosigmoidostomy	Hyperchloremic acidosis

FLUIDS AND ELECTROLYTE ABNORMALITIES

Uretersigmoidostomy is associated with	Hypokalemic hypochloremic metabolic acidosis
Plasma bicarbonate is decreased in	Compensated respiratory alkalosis
2 year boy, fever 3 days, responded to paracetamol, acute renal failure, marked acidosis and encephalopathy. Plenty of oxalate crystals in urine. blood anion gap and osmol gap increased	Diethyl glycol poisoning
In post burns patient	Hyperkalemic acidosis
Change seen in metabolic acidosis	Decreased K+ excretion
50 kg man, metabolic acidosis and pH 7.05. pCO2 12 mm Hg,pO2 108 mm Hg,HCO3 14 mEq/L, approximate quantity of sodium bicarbonate that he should receive in half hour	250 mEq
In metabolic acidosis, kidneys excrete through urine	More ammonia
Hyperventilation occurs as a swift response to	Metabolic acidosis
Kussmaul breathing is seen in	Metabolic acidosis
Kussmaul breathing is also seen in	*Pontine lesion*
Blindness and Metabolic acidosis	Methyl alcohol
Hyperventilation is compensatory mechanism of	Metabolic acidosis

METABOLIC ALKALOSIS

Causes of metabolic alkalosis	Bartter syndrome, Thiazide diuretic therapy, Recurrent vomiting, Cushing disease
MC cause of metabolic alkalosis	Diuretics
Metabolic alkalosis associated with	Hypokalemia
In metabolic alkalosis, true about excretion in urine	Less ammonia
20 mEq of potassium chloride in 500 ml of 5% dextrose solution is given IV in	Metabolic alkalosis
A child with repeated vomiting developed metabolic alkalosis	IV normal saline and potassium
Continuous episode of vomiting may lead to	Decreased K+, decreased Cl- alkalosis
Excessive vomiting cause	Hypokalemia
NOT seen in persistent vomiting	Decreased K+ in urine

RESPIRATORY ACIDOSIS

Low pH, high pCO2 and normal bicarbonate levels	Respiratory acidosis
Respiratory acidosis is biochemically diagnosed by	Increase in PCO2 and decrease in blood pH
PCO2 = 50 mm Hg,PaCO2 = 60 mm Hg, pH 7.1 HCO3- 19 mmol/L	Respiratory acidosis
Respiratory acidosis	Fibrosing alveolitis, Myasthenia gravis, External intercostal muscle paralysis
Respiratory acidosis is seen in	Emphysema
Respiratory acidosis	Increased PCO2 with fall of blood pH
Respiratory acidosis	Diazepam poisoning
Major buffer in respiratory acidosis	Bicarbonate
Synthesis of ammonia is increased in	*Respiratory acidosis*

RESPIRATORY ALKALOSIS

pH 7.46 pCO2 30 mm Hg. patient has partially compensated primary	Respiratory alkalosis
Plasma bicarbonate in Respiratory alkalosis	Low/Normal

ENTERAL NUTRITION

Enteral nutrition reduces	*Infectious complications in critically ill patients*
Souttar tube	*Esophageal feeding tube*
Length of feeding tube to be inserted for transpyloric feeding is measured from	Earlobe to umbilicus
Ramesh met with RTA and in deep coma for 15 days. most suitable route of administration of proteins and calories	Jejunostomy tube feeding
Contraindication of enteral nutrition	Intestinal obstruction, Severe pancreatitis, Severe diarrhea, Intestinal fistula
A patient undergoes prolonged surgery for chronic pancreatitis. Preferred route of supplementary nutrition	Feeding jejunostomy
Disadvantage of elemental diets in children	Hypertonic dehydration, Dumping syndrome
NOT available for TPN but available by enteral nutrition	Dietary fibre, glutamine, short and medium chain fatty acids
Recognized frequent complication about enteral feeding	Diarrhea, Aspiration pneumonitis

TOTAL PARENTERAL NUTRITION

FEATURES OF TPN

65 kg with severe burns in catabolic state. He requires 40 kcal/kg/per day and 2 gm/kg/ day protein. He is given 20 % glucose and 4.25 gm protein. If 3000 ml of solution infused per day	Both protein and calories would be adequate
Post operative patient, peritonitis, massive contamination because of duodenal leak	TPN
NOT an indication of TPN	Chronic liver failure, Sepsis, Pancreatitis
In IV hyper alimentation we give	Fat, amino acids, dextrose
TPN	Lipids provide 20% of energy
NOT included in TPN	Fibers
Best vein for TPN	Subclavian vein
Monitored daily in TPN	Weight, CBC, electrolytes, BUN

COMPLICATIONS OF TPN

Essential fatty acid deficiency is seen	Total parenteral nutrition
Mineral commonly deficient in TPN	*Zinc*
TPN may be complicated by	Hypercalcemia
Weight gain after TPN	7 days
In patients depending entirely on TPN, weight loss per day	150 gm
Complication of TPN	Hyperglycemia, Hypokalemia, Azotemia
Important adverse effect of TPN	Hyperglycemia
Complications of TPN (Refeeding syndrome)	Hypokalemia, hypomagnesemia, hypophosphatemia, hyperglycemia
TPN is associated with	Hyperammonemia
A patient of TPN for 20 days present with weakness, vertigo and convulsion	Hypomagnesemia
Significant unintentional weight loss over 6 months means weight loss more than how much percentage of body weight	5%
NOT a complication of TPN	Congestive cardiac failure
NOT a complication of TPN	Neutrophil dysfunction
Metabolic disorder NOT seen with TPN	Hyperkalemia

SHOCK

GENERAL FEATURES OF SHOCK

Best method to assess intake of fluid in poly trauma. Fluid replacement in trauma is determined by	Urine output
Shock is clinically assessed by	Urine output
Best parameter for assessing shock	Urine output
Heart rate in Shock	Increases
MC type of shock	Hypovolemic shock

ANAPHYLACTIC SHOCK

Drug of choice in anaphylactic shock	IV adrenaline
NOT to be initial priority in anaphylaxis with BP 80/65	Crystalloids
Lady developed breathlessness, pruritis, utricaria, 1 hour after taking NSAIDS for headache and rales. BP 80/50.NOT done	Crystalloids

HYPOVOLEMIC SHOCK

Hypovolemia	**Mild < 20% of total blood volume, moderate 20 – 40%, severe > 40%**
Hypovolemic shock occurs when intravascular volume is decreased by	15-25%
Surface area of burn causing death due to hypovolemic shock	10 – 15%
Triad of low JVP, low blood pressure, raised stroke volume	Hypovolemic shock
Compensatory mechanism in patient with hypovolemic shock	Decreased cutaneous blood flow
BP 70/50 mm hg, serum lactate level 30 mg/100 mL, cardiac output 1.9 L/min, CVP 2 cm H2O	Hypovolemic shock
Child vomiting diarrhea, RR 45/min, HR 130/min, SBP 85 mm hg. capillary refill time 4 seconds	Early compensated hypovolemic shock
An alert 6 month old child is brought with vomiting and diarrhea. RR 45/min, HR 180/min.SBP 85 mm Hg. extremities are cold and mottled. Capillary refilling time is 4 seconds.	Early compensated shock due to hypovolemia
NOT a feature of hypovolemic shock	Bradycardia
NOT a compensation in Hypovolemic shock	Profound vasodilatation
Best fluid of hypovolemic shock	Ringer Lactate

SEPTIC SHOCK

Endotoxin shock is initiated by	Cytokines action
Endotoxin is	Heat stable, weakly antigenic, no enzymatic activity
Endotoxic shock earliest change is due to	Endothelial injury
For which type of Shock, Plasma expanders are given	Endotoxic Shock
Initiating mechanism in endotoxic shock	Endothelial injury, Cytokine release
Septic Shock in children is differentiated from other type of Shock by	Bounding Pulse
Septic shock	Increased cardiac output, decreased total peripheral resistance
In gram negative septicemia, early finding setting before shock	Increased cardiac output and decreased total peripheral resistance
Obtundation is a feature	*Late septic shock*
Cause of renal failure in septicemic shock	Acute tubular necrosis
Most important cause of death in septic shock	Cardiac, Renal
Septicemia, shock in the form of hypotension and low urine output. She was being treated for colonic necrosis. Management	IV fluid only
Drug used to maintain BP in septic shock	Noradrenaline
70 year old man develops pneumonia and septicemia. patient goes into renal failure and has a BP of 70/50 mm Hg. drug used to maintain BP	Norepinephrine
First drug approved by FDA for septic shock	**Recombinant activated protein C**
NOT recommended in septic shock	Rituximab

CARDIOGENIC SHOCK

Lower dose of dopamine in cardiogenic shock will increase	Urine output
Drug given in cardiogenic shock	Dopamine
Drug given in cardiogenic shock	Phentolamine
NOT used in cardiogenic shock	Atropine
NOT true about cardiogenic Shock	Decreased CVP

NEUROGENIC SHOCK

Neurogenic shock is seen in	Anesthesia
Neurogenic shock is associated with	Hypotension + bradycardia

HEMORRHAGIC SHOCK

Blood clot size of a clenched fist is roughly equal to	500 ml
Earliest indicator of concealed acute bleeding	Tachycardia
Blood loss during major surgery assessed by	Suction bottles
Cortisol returns to normal	3 days after hemorrhage
0.5 liter loss of blood in 30 minutes will lead to	Slight increase in heart rate, normal BP
Massive Acute Blood loss is associated with	Increase in reticulocyte count, increase in PCV, increase in neutrophils
NOT seen in massive acute blood loss	Increase in MCV
Femur fracture open with head injury management	IV fluids

MANAGEMENT OF SHOCK

Most important factor in the management of shock	Deficiency of effective circulation
Immediate management of patient with multiple fracture and fluid loss	Ringer lactate
6 month child, IV attempt failed. next step	Intraosseous fluid
Intraosseous administration for age	Less than 6 years
30% loss of blood volume. treatment	IV fluid only
Fluid administration in infant of weight 10 kg	4 ml/Kg/hr
A 70 year old man presenting with acute pain abdomen mass with shock, management	6 units of transfusion, Shift the patient to OT immediately and exploration of abdomen, Laparotomy
Treatment of shock for cold shock which is fluid and dopamine refractory	Epinephrine

ELECTROLYTE IMBALANCE

GENERAL FEATURES OF ELECTROLYTE IMBALANCE

Following fistulous condition give rise to maximum fluid and electrolyte balance	Duodenal
Hypertension with alkalosis	Liddle syndrome
Sweat chloride levels are increased in	Ectodermal dysplasia, G6PD deficiency, hereditary nephrogenic diabetes insipidus
Most abundant anion in blood plasma next to chloride ion	*Bicarbonate*
NOT causing hyperuricemia	Probenecid

INTRAVENOUS FLUIDS

5% dextrose is	*Isotonic*
5% dextrose in 0.9% saline	*Dilutional acidosis*
Amount of K+ in ringer lactate	4 mEq/L
Ringer lactate	Chloride 111, Lactate 29
Ringer lactate does NOT contain	Bicarbonate
Enteric fluid with composition similar to ringer lactate	*Contents of small intestine*
Furasol DA	Used to increase O2 delivery to tissue
Hydroxy ethyl starch	Plasma expander
Plasma expander with oxygen carrying capacity	Perfluorocarbon
Plasma expander having oxygen carrying capacity	Fluorocarbons
How to prepare normal saline from 10% dextrose	80 ml 10% D with 20 ml NS
Hemacel contains	Degraded gelatin
Low molecular weight dextran contraindicated in	Thrombocytopenia

WATER

Water content in infant	75 – 80%
Percentage of water content in total body weight in adult male	More than 60%
Water content of lean body tissue	*71 ml per 100 gram of tissue*
Water balance	*Input - 9 L, reabsorbed - 8800 ml, excreted in stool - 200 ml*
Main source of water loss	**Evaporation from skin and respiratory tract**
Insensible water loss through skin and respiratory tract	500 - 800 ml
Body water content in percentage of body weight is	Fat woman

lowest in	
Commonest cause of water intoxication in surgical patient	Excessive infusion of 5% glucose
Daily loss of water from skin in absence of sweating	200 – 300 ml
Free water clearance	Regulated by ADH
Third space	*Sequestration of fluid in a non functional extracellular space that is beyond osmotic equilibrium with the vascular space*
Post operative third space accumulation is managed by	*Intravenous normal saline*
Changes after intravenous infusion of isotonic saline	*No change in osmolality of ECF*

SODIUM

Sodium content of one liter of isotonic saline	154
Content of sodium in ringer lactate	130
Sodium dependent glucose transport is inhibited by	Phlorhizine
Sodium channels are specifically blocked by	Tetradotoxin
Tetradotoxin blocks	Gamma sodium channel during action potential
Confused state, hypotension, metabolic alkalosis, diagnosis by	Urine Na+
Sodium	Daily intake of 150 mmol of NaCl
Majority of body sodium	Bone

HYPERNATREMIA

MC gastrointestinal cause of hypernatremia	**Diarrhea**
Hypernatremia cause	**Cell shrinkage**
Adipsic hypernatremia	**Defect in thirst mechanism, treatment is water orally**
Proper management of a middle aged woman presenting with serum sodium of 120 mEq/L after successful cholecystectomy	*Free water restriction*
A breast fed child with hypernatremia. His urine sodium is only 70 mEq/L.	Excessive intake of sodium
Correction of hypernatremia should be done over	24 hours

HYPONATREMIA

Concentration of sodium in hyponatremia	**0.5 – 1 meq/L/hr**
Types of hyponatremia	**Type I – edema is present, hypervolemic. Type II – sodium depletion, associated with diarrhea, diuretic abuse, mineralocorticoid deficiency, hypovolemic. Type III – nausea, glucocorticoid deficiency, euvolemic. Type IV – SIADH, euvolemic**
Hyponatremia seen in	Mucoviscoidosis, Diabetes insipidus, Adrenal

	hyperplasia
NOT an electrolyte abnormality in immediate postoperative period	Hyponatremia
NOT an important cause of hyponatremia	Excessive sweating
NOT an important cause of hyponatremia	Prolonged Ryle's tube aspiration
Earliest indicator of sodium loss	Altered sensorium
Hyponatremia	Hyponatremia associated with hyperglycemia has high plasma osmolality, hyponatremia associated with SIADH is normovolemic, NSAIDs increase potency of vasopressin
Euvolemic hyponatremia is seen in	SIADH, Primary Polydipsia, Hypothyroidism
Dilutional hyponatremia is a side effect of	Octreotide, Vincristine, Carbamazepine
Acute hyponatremia in 8 month old infant	Convulsions, Muscle cramps, Vascular collapse
Acute hyponatremia in 8 month old infant does NOT cause	Diarrhea
Pseudohyponatremia is associated with	*Multiple myeloma, hyperlipidemia, severe hyperglycemia*
Hyponatremia in multiple myeloma	Pseudohyponatremia

POTASSIUM

Percentage of plasma K+ in total body potassium	7.6%
Normal requirement of potassium	150 mEq/day
Requirement of potassium in a child	1-2 mEq/kg/day
Highest concentration of potassium is in	Darrow's solution
Highest concentration of potassium in	Colon
Highest concentration of potassium	Rectal fluid
Maximum potassium ion secretion in	Saliva > rectal fluid > pancreatic juice
In immediate post op period body potassium excreted	Excessively
During regulatory volume decrease, many cells will increase	*Efflux of K+*
Potassium balance	Most of the potassium is intracellular, Three quarter of total body potassium is found in skeletal muscle, Intracellular potassium is released into extracellular space in response to severe injury, Alkalosis lead to movement of potassium from extracellular to intracellular fluid compartment
K+ homeostasis in human	An essential electrolyte for different organ function
90% of potassium is excreted in	**Urine**
NOT true about potassium balance	Acidosis leads to movement of potassium from ECF to ICF

HYPERKALEMIA

Spironolactone induced hyperkalemia is potentiated by	NSAID, beta blockers, captopril
Hyperkalemia of ACE inhibitors are aggravated by	Spironolactones
Hyperkalemia is caused by	ACE inhibitors, Cyclosporine, Succinylcholine
NOT a cause of hyperkalemia	Digoxin

Hyperkalemia is NOT caused by	Amphotericin
Elevated serum K+ cause muscle weakness by	*Closure of Na+ channels by depolarisation*
Hyperkalemia causes	*Increased potassium conductance*
Management of hyperkalemia	Intravenous infusion of glucose and insulin, Calcium gluconate, Sodium bicarbonate
Counteracts myocardial effect of potassium without reducing serum potassium level	*Calcium gluconate*
Sodium polystyrene sulphate	**Exchanges Na+ for K+ in gastrointestinal tract, increases fecal excretion of K+**
Kayexalate	**K+ binding resin**
NOT used for treatment of hyperkalemia	Magnesium sulphate
NOT used in treatment of acute hyperkalemia	Potassium binding resins
NOT used for treatment of hyperkalemia	Beta blockers
NOT used for acute hyperkalemia	Potassium exchange resins
Drug NOT used in hyperkalemia without ECG finding	Calcium gluconate
NOT used in hyperkalemia	50 ml of 50% dextrose
Pseudohyperkalemia	**Disorders of cation in RBC**

HYPOKALEMIA

Drugs causing hypokalemia	Amphotericin B, Insulin, Carbenoloxone
Excessive use of Lactulose causes	Hypokalemia
Metabolic changes associated with excess vomiting	Hypokalemia
Electrolyte imbalance causing generalized muscle weakness after barium carbonate ingestion	Hypokalemia
Hypokalemia caused by	Amphotericin B
NOT a feature of Hypokalemia	Diarrhea
A patient of treatment with Amphotericin B with Hypokalemia 2.3 mEq/l. Management	120 mEq/24 hours

MAGNESIUM

Primary site of magnesium reabsorption	*Ascending limb of loop of henle*
Normal serum magnesium	1.4 – 2.4 mEq/L (0.7 – 1 mmol/L)
Mg++ is contraindicated in	Renal failure, intrauterine infection, Premature rupture of membranes

HYPERMAGNESEMIA

Hypermagnesemia is associated with	**Vasodilatation, neuromuscular blockade**
Hypermagnesemia is associated with	*Meconium plug syndrome*

HYPOMAGNESEMIA

Drug causing hypomagnesemia	*Foscarnet*
Causes of hypomagnesemia	*Hyperaldosteronism, malabsoption, alcoholic cirrhosis, short bowel syndrome*
Early sign of hypomagnesemia	Hypotension
Hypomagnesemia due to increased excretion in kidney caused by	Furosemide, Aminoglycosides, Cisplatin
Drug causing hypomagnesemia by increased excretion	Furosemide therapy
NOT a cause of Hypomagnesemia due to increased excretion in kidney	Digitalis

PHOSPHATE

FGF 23	**Phosphatonin**
Normal phosphate	**2.5 – 4.5 mg/dl**
Intestinal phosphate absorption is retarded by	**Aluminium hydroxide**
Renal phosphate excretion is promoted by	**FGF23**
Urinary Phosphoethanolamine	Hypophosphatasia
Hypophosphatasia is associated with	**Decreased Alkaline phosphatase**
Hypophosphatemia	Resolving phases of diabetic ketoacidosis, Respiratory alkalosis/COPD, Chronic alcoholism
Hyperphosphatemia is associated with	*CRF*
Hypophosphatasia is associated with	*Low serum alkaline phosphatase*
NOT a cause of hypophosphatemia	*Acute renal failure*
Refeeding syndrome	**Hypophosphatemia aggravated by glucose infusion**
Phosphate binders	**Calcium acetate, calcium carbonate, sevelamer, lanthanum**